Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
S1	3	US-5789538-$.DID. OR US-6007408-$.DID. OR US-6013453-$.DID.	USPAT	ADJ	ON	2004/07/19 12:41
S2	3	binding site with cellular chromatin	USPAT	ADJ	ON	2004/07/17 15:48
S3	3	S2 not S1	USPAT	ADJ	ON	2004/07/17 15:48
S4	3	US-5306619-$.DID. OR US-6410248-$.DID. OR US-6007988-$.DID.	USPAT	ADJ	ON	2004/07/19 12:44
S5	3	US-5789538-$.DID. OR US-6007408-$.DID. OR US-6013453-$.DID.	USPAT	ADJ	ON	2004/07/19 12:50
S6	23554	chromatin or chromosome or episome or nucleosome	USPAT	ADJ	ON	2004/07/19 12:49
S7	274	S6 with binding site	USPAT	ADJ	ON	2004/07/19 12:49
S8	2	S7 WITH (zinc finger)	USPAT	ADJ	ON	2004/07/19 12:52
S9	90	S7 WITH (protein)	USPAT	ADJ	ON	2004/07/19 12:52
S10	480	bind$ with minor groove	USPAT	ADJ	ON	2004/07/19 13:00
S11	71	S10 with protein	USPAT	ADJ	ON	2004/07/19 13:00
S12	71	S11 not S9	USPAT	ADJ	ON	2004/07/19 13:01
S13	1	S12 with chromatin	USPAT	ADJ	ON	2004/07/19 13:02
S14	10	S10 with zinc finger	USPAT	ADJ	ON	2004/07/19 13:02

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  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *


*CHEMLIST      - Regulated Chemicals Listing
*HCHEMLIST     - Regulated Chemicals Listing with hour-based pricing

  * The files listed above are temporarily unavailable.

FILE 'HOME' ENTERED AT 13:36:35 ON 19 JUL 2004

=> index biosci
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
       BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT,
       CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS, DDFB, DDFU,
       DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 13:36:48 ON 19 JUL 2004

70 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s chromatin (p) bind?
          2   FILE ADISCTI
          1   FILE ADISINSIGHT
          1*  FILE ADISNEWS
        247   FILE AGRICOLA
          1   FILE ANABSTR
         81   FILE AQUASCI
         10   FILE BIOBUSINESS
          0*  FILE BIOCOMMERCE
       6287   FILE BIOSIS
        127*  FILE BIOTECHABS
        127*  FILE BIOTECHDS
       3979*  FILE BIOTECHNO
        405   FILE CABA
       1956   FILE CANCERLIT
       7229   FILE CAPLUS
          8*  FILE CEABA-VTB
          3*  FILE CIN
         53   FILE CONFSCI
          5   FILE CROPB
          3   FILE CROPU
        800   FILE DISSABS
        107   FILE DDFB
         91   FILE DDFU
       1329   FILE DGENE
  25 FILES SEARCHED...
        107   FILE DRUGB
          2   FILE IMSDRUGNEWS
        204   FILE DRUGU
          2   FILE IMSRESEARCH
        128   FILE EMBAL
       5214   FILE EMBASE
       4341*  FILE ESBIOBASE
        575*  FILE FEDRIP
          0*  FILE FOMAD
          0*  FILE FOREGE
          2*  FILE FROSTI
          5*  FILE FSTA
        464   FILE GENBANK
          6   FILE HEALSAFE
        110   FILE IFIPAT
        102   FILE JICST-EPLUS
          3*  FILE KOSMET
       3424   FILE LIFESCI
          0*  FILE MEDICONF
       5940   FILE MEDLINE
         60   FILE NIOSHTIC
         70*  FILE NTIS
          0*  FILE NUTRACEUT
          2   FILE OCEAN
       1051*  FILE PASCAL
          0*  FILE PHARMAML
          1   FILE PHIN
         24   FILE PROMT
  58 FILES SEARCHED...
       5900   FILE SCISEARCH
       2647   FILE TOXCENTER
       1414   FILE USPATFULL
         62   FILE USPAT2
          1   FILE VETU
        107   FILE WPIDS
          2   FILE WPIFV
        107   FILE WPINDEX

  54 FILES HAVE ONE OR MORE ANSWERS,   70 FILES SEARCHED IN STNINDEX

L1    QUE CHROMATIN (P) BIND?

=> file biotechno biosis lifesci medline
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.28       2.49

FILE 'BIOTECHNO' ENTERED AT 13:38:58 ON 19 JUL 2004
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FILE 'BIOSIS' ENTERED AT 13:38:58 ON 19 JUL 2004
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FILE 'LIFESCI' ENTERED AT 13:38:58 ON 19 JUL 2004
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FILE 'MEDLINE' ENTERED AT 13:38:58 ON 19 JUL 2004

=> s l1
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CHROMATIN (P) BIND?'
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L2 19630 L1

=> s l2 and (zinc (w) finger) or zfp
L3 784 L2 AND (ZINC (W) FINGER) OR ZFP

=> s l3 and intracell?
L4 14 L3 AND INTRACELL?

=> d l4 bib ab 1-14

L4 ANSWER 1 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 2003:37272164 BIOTECHNO
TI The rise of DNA methylation and the importance of ***chromatin*** on multidrug resistance in cancer
AU Baker E.K.; El-Osta A.
CS A. El-Osta, Alfred Med. Res./Education Precinct, Baker Medical Research Institute, Epigenetics in Hum. Hlth./Dis. Lab., Commercial Road, Prahran, Vic. 3181, Australia.
E-mail: assam.el-osta@baker.edu.au
SO Experimental Cell Research, (01 NOV 2003), 290/2 (177-194), 197 reference(s)
CODEN: ECREAL ISSN: 0014-4827
DT Journal; General Review
CY United States
LA English
SL English
AB In recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces ***intracellular*** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has been unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation or genetic amplification is by no means the only mechanism of activation. Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 ***chromatin*** influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered ***ZFP*** 's (***zinc*** ***finger*** proteins). .COPYRGT. 2003 Elsevier Inc. All rights reserved.

L4 ANSWER 2 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 2002:34429757 BIOTECHNO
TI Nitrate and the GATA factor AreA are necessary for in vivo ***binding*** of NirA, the pathway-specific transcriptional activator of Aspergillus nidulans
AU Narendja F.; Goller S.P.; Wolschek M.; Strauss J.
CS J. Strauss, Zentrum fur Angewandte Genetik, Univ. of Agricultural Sci. Vienna, Vienna, Austria.
E-mail: jstrauss@edv2.boku.ac.at
SO Molecular Microbiology, (2002), 44/2 (573-583), 53 reference(s)
CODEN: MOMIEE ISSN: 0950-382X
DT Journal; Article
CY United Kingdom
LA English
SL English
AB In Aspergillus nidulans, the genes coding for nitrate reductase (niaD) and nitrite reductase (niiA), are transcribed divergently from a common promoter region of 1200 basepairs. We have previously characterized the relevant cis-acting elements for the two synergistically acting transcriptional activators NirA and AreA. We have further shown that AreA is constitutively bound to a central cluster of four GATA sites, and is involved in opening the ***chromatin*** structure over the promoter region thus making additional cis-acting ***binding*** sites accessible. Here we show that the asymmetric mode of NirA-DNA interaction determined in vitro is also found in vivo. ***Binding*** of the NirA transactivator is not constitutive as in other binuclear C6-Zn.sup.2.sup.+-cluster proteins but depends on nitrate induction and, additionally, on the presence of a wild-type areA allele. Dissecting the role of AreA further, we found that it is required for ***intracellular*** nitrate accumulation and therefore could indirectly exert its effect on NirA via inducer exclusion. We have tested this possibility in a strain accumulating nitrate in the absence of areA. We found that in such a strain the ***intracellular*** presence of inducer is not sufficient to promote either ***chromatin*** rearrangement or NirA ***binding*** , implying that both processes are directly dependent on AreA.

L4 ANSWER 3 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28240528 BIOTECHNO
TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage
AU Masson M.; Niedergang C.; Schreiber V.; Muller S.; Menissier-De Murcia J.; De Murcia G.
CS G. De Murcia, Ecole Sup. Biotechnol. de Strasbourg, UPR 9003 du Ctr. Natl. Rech. Sci., Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France.
E-mail: demurcia@esbs.u-strasbg.fr
SO Molecular and Cellular Biology, (1998), 18/6 (3563-3571), 58 reference(s)
CODEN: MCEBD4 ISSN: 0270-7306
DT Journal; Article
CY United States
LA English
SL English
AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** - ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribosyl)ation of nuclear proteins involved in ***chromatin*** architecture and DNA

metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PARP activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase .beta., our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

L4 ANSWER 4 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:13460 BIOSIS
DN PREV200400017673
TI The rise of DNA methylation and the importance of chromatin on multidrug resistance in cancer.
AU Baker, Emma K.; El-Osta, Assam [Reprint Author]
CS Alfred Medical Research and Education Precinct (AMREP), Baker Medical Research Institute, Epigenetics in Human Health and Disease Laboratory, Commercial Road, Second Floor, Prahran, VIC, 3181, Australia
assam.el-osta@baker.edu.au
SO Experimental Cell Research, (November 1 2003) Vol. 290, No. 2, pp. 177-194. print.
ISSN: 0014-4827 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 24 Dec 2003
Last Updated on STN: 24 Dec 2003
AB In recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces ***intracellular*** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has been unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation or genetic amplification is by no means the only mechanism of activation. Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 chromatin influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered ***ZFP*** 's (zinc finger proteins).

L4 ANSWER 5 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:174793 BIOSIS
DN PREV200200174793
TI Retrovirally expressed metal response element-binding transcription factor-1 normalizes metallothionein-1 gene expression and protects cells against zinc, but not cadmium, toxicity.
AU Solis, Willy A.; Childs, Nicole L.; Weedon, Michael N.; He, Lei; Nebert, Daniel W.; Dalton, Timothy P. [Reprint author]
CS Department of Environmental Health, University of Cincinnati Medical Center, Cincinnati, OH, 45267-0056, USA
tim.dalton@uc.edu
SO Toxicology and Applied Pharmacology, (January 15, 2002) Vol. 178, No. 2, pp. 93-101. print.
CODEN: TXAPA9. ISSN: 0041-008X.
DT Article
LA English
ED Entered STN: 6 Mar 2002
Last Updated on STN: 6 Mar 2002
AB Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys2-His2 class of ***zinc*** - ***finger*** transcription factors, is best known for its robust transcriptional regulation of mammalian metallothionein (MT) genes. MTF1 is also believed to play a generalized role in regulating genes involved in protection against heavy metals and oxidative stress. MTF1 ***binding*** to MRE motifs is regulated by changes in ***intracellular*** zinc (Zn2+) concentration. Molecular dissection of MTF1 has been hindered by its high constitutive trans-activity following transient transfection and the failure of these systems to examine genes packaged in native ***chromatin*** . In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTF1 into mouse Mtf1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTF1 retrovirally transduced dko7 cells (MTF1dko7) possess levels of inducible MTF1-MRE ***binding*** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTF1 ***binding*** could be modulated over a 20-fold range by varying the concentration of Zn2+ present in the culture medium. The dko7 cells exhibited no change in Mt1 gene expression upon Zn2+ or cadmium (Cd2+) treatment; in contrast, in MTF1dko7 cells, Zn2+ or Cd2+ induced MT1 mRNA accumulation in a dose-dependent manner. Interestingly, MTF1dko7 cells showed resistance to Zn2+ toxicity, but negligible resistance to Cd2+. Concomitantly, MT1 protein levels in MTF1dko7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn2+, but not with Cd2+. Together, our studies suggest that MTF1-mediated regulation of gene expression is sufficient to protect cells against Zn2+ toxicity and may be necessary but not sufficient to protect cells against Cd2+ toxicity.

L4 ANSWER 6 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 1998:296836 BIOSIS
DN PREV199800296836
TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage.
AU Masson, Murielle; Niedergang, Claude; Schreiber, Valerie; Muller, Sylviane; Menissier-De Murcia, Josiane; De Murcia, Gilbert [Reprint author]
CS Ecole Superieure Biotechnol. Strasbourg, UPR 9003 Cent. Natl. Rech. Sci., Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France
SO Molecular and Cellular Biology, (June, 1998) Vol. 18, No. 6, pp. 3563-3571. print.
CODEN: MCEBD4. ISSN: 0270-7306.
DT Article
LA English
ED Entered STN: 15 Jul 1998
Last Updated on STN: 15 Jul 1998
AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** - ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribosyl)ation of nuclear proteins involved in ***chromatin*** architecture and DNA metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PAR-P activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta, our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

L4 ANSWER 7 OF 14 LIFESCI COPYRIGHT 2004 CSA on STN
AN 2004:54042 LIFESCI
TI Attenuation of HIV-1 Replication in Primary Human Cells with a Designed ***Zinc*** ***Finger*** Transcription Factor
AU Segal, D.J.; Goncalves, J.; Eberhardy, S.; Swan, C.H.; Torbett, B.E.; Li, X.; Barbas, C.F.
CS The Skaggs Institute for Chemical Biology and the Departments of Molecular Biology and Chemistry
SO Journal of Biological Chemistry [J. Biol. Chem.], (20040409) vol. 279, no. 15, pp. 14509-14519.
ISSN: 0021-9258.
DT Journal
FS N; V
LA English
SL English
AB Small molecule inhibitors of human immunodeficiency virus, type 1 (HIV-1) have been extremely successful but are associated with a myriad of undesirable effects and require lifelong daily dosing. In this study we explore an alternative approach, that of inducing ***intracellular*** immunity using designed, ***zinc*** ***finger*** -based transcription factors. Three transcriptional repression proteins were engineered to ***bind*** sites in the HIV-1 promoter that were expected to be both accessible in ***chromatin*** structure and highly conserved in sequence structure among the various HIV-1 subgroups. Transient transfection assays identified one factor, KRAB-HLTR3, as being able to achieve 100-fold repression of an HIV-1 promoter. Specificity of repression was demonstrated by the lack of repression of other promoters. This factor was further shown to repress the replication of several HIV-1 viral strains 10- to 100-fold in T-cell lines and primary human peripheral blood mononuclear cells. Repression was observed for at least 18 days with no significant cytotoxicity. Stable T-cell lines expressing the factor also do not show obvious signs of cytotoxicity. These characteristics present KRAB-HLTR3 as an attractive candidate for development in an ***intracellular*** immunization strategy for anti-HIV-1 therapy.

L4 ANSWER 8 OF 14 LIFESCI COPYRIGHT 2004 CSA on STN
AN 2002:44734 LIFESCI
TI Retrovirally Expressed Metal Response Element-Binding Transcription Factor-1 Normalizes Metallothionein-1 Gene Expression and Protects Cells against Zinc, but Not Cadmium, Toxicity
AU Solis, W.A.; Childs, N.L.; Weedon, M.N.; He, L.; Nebert, D.W.; Dalton, T.P.*
CS Center for Environmental Genetics, Department of Environmental Health, University of Cincinnati Medical Center, Cincinnati, Ohio, 45267-0056; E-mail: tim.dalton@uc.edu
SO Toxicology and Applied Pharmacology [Toxicol. Appl. Pharmacol.], (20020115) vol. 178, no. 2, pp. 93-101.
ISSN: 0041-008X.
DT Journal
FS X
LA English
SL English
AB Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys sub(2)-His sub(2) class of ***zinc*** - ***finger*** transcription factors, is best known for its robust transcriptional regulation of mammalian metallothionein (MT) genes. MTF1 ***binding*** to MRE motifs is regulated by changes in ***intracellular*** zinc (Zn super(2+)) concentration. Molecular dissection of MTF1 has been hindered by its high constitutive trans -activity following transient transfection and the failure of these systems to examine genes packaged in native ***chromatin*** . In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTF1 into mouse Mtf1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTF1 retrovirally transduced dko7 cells (MTF1dko7) possess levels of inducible MTF1-MRE ***binding*** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTF1 ***binding*** could be modulated over a 20-fold range by varying the concentration of Zn super(2+) present in the culture medium. The dko7 cells exhibited no change in Mt1 gene expression upon Zn super(2+) or

cadmium (Cd super(2+)) treatment; in contrast, in MTF1dko7 cells, Zn super(2+) or Cd super(2+) induced MT1 mRNA accumulation in a dose-dependent manner. Interestingly, MTF1dko7 cells showed resistance to Zn super(2+) toxicity, but negligible resistance to Cd super(2+). Concomitantly, MT1 protein levels in MTF1dko7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn super(2+), but not with Cd super(2+). Together, our studies suggest that MTF1-mediated regulation of gene expression is sufficient to protect cells against Zn super(2+) toxicity and may be necessary but not sufficient to protect cells against Cd super(2+) toxicity. [copy]2002 Elsevier Science (USA).

L4 ANSWER 9 OF 14 LIFESCI COPYRIGHT 2004 CSA on STN
AN 1998:107059 LIFESCI
TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage
AU Masson, M.; Niedergang, C.; Schreiber, V.; Muller, S.; Menissier-de Murcia, J.; De Murcia, G.
CS Ecole Superieure de Biotechnologie de Strasbourg, UPR 9003 du Centre National de la Recherche Scientifique, Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France
SO Mol. Cell. Biol., (19980600) vol. 18, no. 6, pp. 3563-3571. ISSN: 0270-7306.
DT Journal
FS N
LA English
SL English
AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** -***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribosyl)ation of nuclear proteins involved in ***chromatin*** architecture and DNA metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PARP activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta , our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

L4 ANSWER 10 OF 14 MEDLINE on STN
AN 2004172762 MEDLINE
DN PubMed ID: 14734553
TI Attenuation of HIV-1 replication in primary human cells with a designed ***zinc*** ***finger*** transcription factor.
AU Segal David J; Goncalves Joao; Eberhardy Scott; Swan Christina H; Torbett Bruce E; Li Xuelin; Barbas Carlos F 3rd
CS The Skaggs Institute for Chemical Biology and the Departments of Molecular Biology and Chemistry, The Scripps Research Institute, La Jolla, California 92037, USA.
NC GM065059 (NIGMS)
R01 AI49165 (NIAID)
SO Journal of biological chemistry, (2004 Apr 9) 279 (15) 14509-19. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AY518586; GENBANK-AY518587; GENBANK-AY518588
EM 200406
ED Entered STN: 20040407
Last Updated on STN: 20040602
Entered Medline: 20040601
AB Small molecule inhibitors of human immunodeficiency virus, type 1 (HIV-1) have been extremely successful but are associated with a myriad of undesirable effects and require lifelong daily dosing. In this study we explore an alternative approach, that of inducing ***intracellular*** immunity using designed, ***zinc*** ***finger*** -based transcription factors. Three transcriptional repression proteins were engineered to ***bind*** sites in the HIV-1 promoter that were expected to be both accessible in ***chromatin*** structure and highly conserved in sequence structure among the various HIV-1 subgroups. Transient transfection assays identified one factor, KRAB-HLTR3, as being able to achieve 100-fold repression of an HIV-1 promoter. Specificity of repression was demonstrated by the lack of repression of other promoters. This factor was further shown to repress the replication of several HIV-1 viral strains 10- to 100-fold in T-cell lines and primary human peripheral blood mononuclear cells. Repression was observed for at least 18 days with no significant cytotoxicity. Stable T-cell lines expressing the factor also do not show obvious signs of cytotoxicity. These characteristics present KRAB-HLTR3 as an attractive candidate for development in an ***intracellular*** immunization strategy for anti-HIV-1 therapy.

L4 ANSWER 11 OF 14 MEDLINE on STN
AN 2003491182 MEDLINE
DN PubMed ID: 14567978
TI The rise of DNA methylation and the importance of chromatin on multidrug resistance in cancer.
AU Baker Emma K; El-Osta Assam
CS The Alfred Medical Research and Education Precinct, Baker Medical Research Institute, Epigenetics in Human Health and Disease Laboratory, Second Floor, Commercial Road, Prahran, Victoria 3181, Australia.
SO Experimental cell research, (2003 Nov 1) 290 (2) 177-94. Ref: 197 Journal code: 0373226. ISSN: 0014-4827.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals

EM 200312
ED Entered STN: 20031022
Last Updated on STN: 20031219
Entered Medline: 20031202
AB In recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces ***intracellular*** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has been unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation or genetic amplification is by no means the only mechanism of activation. Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 chromatin influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered ***ZFP*** 's (zinc finger proteins).

L4 ANSWER 12 OF 14 MEDLINE on STN
AN 2002120545 MEDLINE
DN PubMed ID: 11814329
TI Retrovirally expressed metal response element-binding transcription factor-1 normalizes metallothionein-1 gene expression and protects cells against zinc, but not cadmium, toxicity.
AU Solis Willy A; Childs Nicole L; Weedon Michael N; He Lei; Nebert Daniel W; Dalton Timothy P
CS Center for Environmental Genetics, University of Cincinnati Medical Center, Cincinnati, Ohio 45267-0056, USA.
NC P30 ES06096 (NIEHS)
R01 AG09235 (NIA)
R01 ES10416 (NIEHS)
SO Toxicology and applied pharmacology, (2002 Jan 15) 178 (2) 93-101.
Journal code: 0416575. ISSN: 0041-008X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200203
ED Entered STN: 20020222
Last Updated on STN: 20020308
Entered Medline: 20020307
AB Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys2-His2 class of ***zinc*** - ***finger*** transcription factors, is best known for its robust transcriptional regulation of mammalian metallothionein (MT) genes. MTF1 is also believed to play a generalized role in regulating genes involved in protection against heavy metals and oxidative stress. MTF1 ***binding*** to MRE motifs is regulated by changes in ***intracellular*** zinc (Zn(2+)) concentration. Molecular dissection of MTF1 has been hindered by its high constitutive trans-activity following transient transfection and the failure of these systems to examine genes packaged in native ***chromatin*** . In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTF1 into mouse Mtf1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTF1 retrovirally transduced dko7 cells (MTF1dko7) possess levels of inducible MTF1-MRE ***binding*** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTF1 ***binding*** could be modulated over a 20-fold range by varying the concentration of Zn(2+) present in the culture medium. The dko7 cells exhibited no change in Mt1 gene expression upon Zn(2+) or cadmium (Cd(2+)) treatment; in contrast, in MTF1dko7 cells, Zn(2+) or Cd(2+) induced MT1 mRNA accumulation in a dose-dependent manner. Interestingly, MTF1dko7 cells showed resistance to Zn(2+) toxicity, but negligible resistance to Cd(2+). Concomitantly, MT1 protein levels in MTF1dko7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn(2+), but not with Cd(2+). Together, our studies suggest that MTF1-mediated regulation of gene expression is sufficient to protect cells against Zn(2+) toxicity and may be necessary but not sufficient to protect cells against Cd(2+) toxicity.
2002 Elsevier Science (USA).

L4 ANSWER 13 OF 14 MEDLINE on STN
AN 2001539383 MEDLINE
DN PubMed ID: 11586467
TI Transcriptional regulation in hepatic stellate cells.
AU Eng F J; Friedman S L
CS Division of Liver Diseases, Department of Medicine, Mount Sinai School of Medicine, 1425 Madison Ave., New York, NY 10029, USA.
SO Seminars in liver disease, (2001 Aug) 21 (3) 385-95. Ref: 81
Journal code: 8110297. ISSN: 0272-8087.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200111
ED Entered STN: 20011008
Last Updated on STN: 20011105
Entered Medline: 20011101
AB Modulation of gene expression through altered transcription regulates stellate cell behavior in normal liver and following hepatic injury. Transcription factors are generally classified according to conserved motifs within either the activation- or DNA- ***binding*** domains of the molecules. Transcriptional activity in stellate cells represents a delicate fine tuning of multiple inputs. Activities of these transcription factors are modified by their ***intracellular*** localization, rate and pathway of degradation, oligomerization, and interactions with heterologous factors and ***chromatin*** , as well as by posttranslational modifications, including phosphorylation, glycosylation, and acetylation. General paradigms of transcriptional

control are increasingly being validated in hepatic stellate cells, particularly involving the transcription factors CCAAT/enhancer- ***binding*** proteins, c-myb, CREB, nuclear factor kappaB, peroxisome proliferator-activated receptor, and Kruppel-like ***zinc*** ***finger*** factors. Although there are no simple rules that govern mechanisms of transcriptional regulation in stellate cells, continued advances will yield new insights into their role in normal liver homeostasis and in the response to injury.

L4 ANSWER 14 OF 14 MEDLINE on STN
AN 1998252943 MEDLINE
DN PubMed ID: 9584196
TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage.
AU Masson M; Niedergang C; Schreiber V; Muller S; Menissier-de Murcia J; de Murcia G
CS UPR 9003 du Centre National de la Recherche Scientifique, Cancerogenese et Mutagenese Moleculaire et Structurale, Ecole Superieure de Biotechnologie de Strasbourg, 67400 Illkirch-Graffenstaden, France.
SO Molecular and cellular biology, (1998 Jun) 18 (6) 3563-71.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199806
ED Entered STN: 19980625
Last Updated on STN: 19980625
Entered Medline: 19980617
AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** - ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribosyl)ation of nuclear proteins involved in ***chromatin*** architecture and DNA metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PARP activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta, our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

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=> log h
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    22.10      24.59

 SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 13:40:05 ON 19 JUL 2004

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LOGINID:SSSPTA1653RAW

PASSWORD:
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AT 13:44:54 ON 19 JUL 2004
FILE 'BIOTECHNO' ENTERED AT 13:44:54 ON 19 JUL 2004
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FILE 'MEDLINE' ENTERED AT 13:44:54 ON 19 JUL 2004

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    22.10      24.59

=> s chromatin and binding (w) site and protein
L5        1608 CHROMATIN AND BINDING (W) SITE AND PROTEIN

=> dup rem l5
PROCESSING IS APPROXIMATELY  75% COMPLETE FOR L5
PROCESSING COMPLETED FOR L5
L6         842 DUP REM L5 (766 DUPLICATES REMOVED)

=> s l6 and zinc (w) finger
L7          39 L6 AND ZINC (W) FINGER

=> d his

     (FILE 'HOME' ENTERED AT 13:36:35 ON 19 JUL 2004)

     INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS,
     DDFB, DDFU, DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 13:36:48 ON 19 JUL
     2004
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              2   FILE ADISCTI
              1   FILE ADISINSIGHT
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   1   FILE ANABSTR
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  10   FILE BIOBUSINESS
   0*  FILE BIOCOMMERCE
6287   FILE BIOSIS
 127*  FILE BIOTECHABS
 127*  FILE BIOTECHDS
3979*  FILE BIOTECHNO
 405   FILE CABA
1956   FILE CANCERLIT
7229   FILE CAPLUS
   8*  FILE CEABA-VTB
   3*  FILE CIN
  53   FILE CONFSCI
   5   FILE CROPB
   3   FILE CROPU
 800   FILE DISSABS
 107   FILE DDFB
  91   FILE DDFU
1329   FILE DGENE
 107   FILE DRUGB
   2   FILE IMSDRUGNEWS
 204   FILE DRUGU
   2   FILE IMSRESEARCH
 128   FILE EMBAL
5214   FILE EMBASE
4341*  FILE ESBIOBASE
 575*  FILE FEDRIP
   0*  FILE FOMAD
   0*  FILE FOREGE
   2*  FILE FROSTI
   5*  FILE FSTA
 464   FILE GENBANK
   6   FILE HEALSAFE
 110   FILE IFIPAT
 102   FILE JICST-EPLUS
   3*  FILE KOSMET
3424   FILE LIFESCI
   0*  FILE MEDICONF
5940   FILE MEDLINE
  60   FILE NIOSHTIC
  70*  FILE NTIS
   0*  FILE NUTRACEUT
   2   FILE OCEAN
1051*  FILE PASCAL
   0*  FILE PHARMAML
   1   FILE PHIN
  24   FILE PROMT
5900   FILE SCISEARCH
2647   FILE TOXCENTER
1414   FILE USPATFULL
  62   FILE USPAT2
   1   FILE VETU
 107   FILE WPIDS
   2   FILE WPIFV
 107   FILE WPINDEX
L1          QUE CHROMATIN (P) BIND?
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    FILE 'BIOTECHNO, BIOSIS, LIFESCI, MEDLINE' ENTERED AT 13:38:58 ON 19 JUL
    2004
L2        19630 S L1
L3          784 S L2 AND (ZINC (W) FINGER) OR ZFP
L4           14 S L3 AND INTRACELL?
L5         1608 S CHROMATIN AND BINDING (W) SITE AND PROTEIN
L6          842 DUP REM L5 (766 DUPLICATES REMOVED)
L7           39 S L6 AND ZINC (W) FINGER

=> s l7 not l4
L8           39 L7 NOT L4

=> s l8 and py<=2000
   3 FILES SEARCHED...
L9           15 L8 AND PY<=2000

=> d l9 1-15 bib ab

L9    ANSWER 1 OF 15  BIOTECHNO  COPYRIGHT 2004 Elsevier Science B.V. on STN
AN    2000:30808610   BIOTECHNO
TI    Synthetic   ***zinc***     ***finger***   transcription factor action at
      an endogenous chromosomal site: Activation of the human erythropoietin
      gene
AU    Zhang L.; Spratt S.K.; Liu Q.; Johnstone B.; Qi H.; Raschke E.E.;
      Jamieson A.C.; Rebar E.J.; Wolffe A.P.; Case C.C.
CS    A.P. Wolffe, Sangamo BioSciences Inc., Point Richmond Tech. Center, 561
      Canal Blvd., Richmond, CA 94804, United States.
      E-mail: awolffe@sangamo.com
SO    Journal of Biological Chemistry,   ***(27 OCT 2000)***  , 275/43
      (33850-33860), 39 reference(s)
      CODEN: JBCHA3  ISSN: 0021-9258
DT    Journal; Article
CY    United States
LA    English
SL    English
AB    We have targeted the activation of an endogenous chromosomal locus
      including the human erythropoietin gene using synthetic transcription
      factors. These transcription factors are targeted to particular DNA
      sequences in the 5'-flanking region of the erythropoietin gene through
      engineering of a   ***zinc***     ***finger***   DNA binding domain. The
      DNA binding domain is linked to a VP16 transcriptional activation domain.
      We find that these synthetic transcription factors invariably activate
      transiently transfected templates in which sequences within the 5' flank
      of the erythropoietin gene are fused to a luciferase reporter. The
      efficiency of activation under these circumstances at a defined site is
      dependent on DNA binding affinity. In contrast, only a subset of these
      same   ***zinc***     ***finger***   proteins is able to activate the
      endogenous chromosomal locus. The activity of these proteins is
      influenced by their capacity to gain access to their recognition elements
      within the   ***chromatin***   infrastructure.   ***Zinc***
        ***finger***   transcription factors will provide a powerful tool to
      probe the determinants of   ***chromatin***   accessibility and
      remodeling within endogenous chromosomal loci.
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L9 ANSWER 2 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 2000:30736881 BIOTECHNO
TI An Ikaros-containing ***chromatin*** -remodeling complex in adult-type erythroid cells
AU O'Neill D.W.; Schoetz S.S.; Lopez R.A.; Castle M.; Rabinowitz L.; Shor E.; Krawchuk D.; Goll M.G.; Renz M.; Seelig H.-P.; Han S.; Seong R.H.; Park S.D.; Agalioti T.; Munshi N.; Thanos D.; Erdjument-Bromage H.; Tempst P.; Bank A.
CS A. Bank, Dept. of Genetics and Development, Hammer Health Sciences, 701 West 168th Street, New York, NY 10032, United States.
E-mail: bank@cuccfa.ccc.columbia.edu
SO Molecular and Cellular Biology, (***2000***), 20/20 (7572-7582), 59 reference(s)
CODEN: MCEBD4 ISSN: 0270-7306
DT Journal; Article
CY United States
LA English
SL English
AB We have previously described a SWI/SNF-related ***protein*** complex (PYR complex) that is restricted to definitive (adult-type) hematopoietic cells and that specifically binds DNA sequences containing long stretches of pyrimidines. Deletion of an intergenic DNA- ***binding*** ***site*** for this complex from a human .beta.-globin locus construct results in delayed human .gamma.- to .beta.-globin switching in transgenic mice, suggesting that the PYR complex acts to facilitate the switch. We now show that PYR complex DNA-binding activity also copurifies with subunits of a second type of ***chromatin*** -remodeling complex, nucleosome-remodeling deacetylase (NuRD), that has been shown to have both nucleosome-remodeling and histone deacetylase activities. Gel supershift assays using antibodies to the ATPase-helicase subunit of the NuRD complex, Mi-2 (CHD4), confirm that Mi-2 is a component of the PYR complex. In addition, we show that the hematopoietic cell-restricted ***zinc*** ***finger*** ***protein*** Ikaros copurifies with PYR complex DNA-binding activity and that antibodies to Ikaros also supershift the complex. We also show that NuRD and SWI/SNF components coimmunopurify with each other as well as with Ikaros. Competition gel shift experiments using partially purified PYR complex and recombinant Ikaros ***protein*** indicate that Ikaros functions as a DNA-binding subunit of the PYR complex. Our results suggest that Ikaros targets two types of ***chromatin*** -remodeling factors - activators (SWI/SNF) and repressors (NuRD) - in a single complex (PYR complex) to the .beta.-globin locus in adult erythroid cells. At the time of the switch from fetal to adult globin production, the PYR complex is assembled and may function to repress .gamma.-globin gene expression and facilitate .beta.- to .beta.-globin switching.

L9 ANSWER 3 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 2000:30710842 BIOTECHNO
TI Transcriptional activation by the PHD finger is inhibited through an adjacent leucine zipper that binds 14-3-3 proteins
AU Halbach T.; Scheer N.; Werr W.
CS W. Werr, Institut fur Entwicklungsbiologie, Universitat zu Koln, Gyrhofstrabe 17, 50923 Koln, Germany.
E-mail: w.werr@uni-koeln.de
SO Nucleic Acids Research, ***(15 SEP 2000)*** , 28/18 (3542-3550), 50 reference(s)
CODEN: NARHAD ISSN: 0305-1048
DT Journal; Article
CY United Kingdom
LA English
SL English
AB The PHD finger, a Cys.sub.4-His-Cys.sub.3 ***zinc*** ***finger*** , is found in many regulatory proteins from plants or animals which are frequently associated with ***chromatin*** -mediated transcriptional regulation. We show here that the PHD finger activates transcription in yeast, plant and animal cells. In plant homeodomain transcription factors the PHD finger is combined with an upstream leucine zipper. Both domains together form a highly conserved 180 amino acid region called the ZIP/PHDf motif and transcriptional activity of the PHD finger is masked when embedded in this motif. Our results indicate that the ZIP/PHDf domain is a potential regulatory domain of PHDf-HD proteins. The leucine zipper upstream of the PHD finger interacts with 14-3-3GF14.mu. from Arabidopsis thaliana and 14-3-3GF14-12 from maize via a leucine zipper conserved in helix 4 of various 14-3-3 proteins from plants and animals. PHD-type plant homeodomain proteins consequently may represent potential targets of 14-3-3 signalling.

L9 ANSWER 4 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 2000:30329448 BIOTECHNO
TI Maternal-specific footprints at putative CTCF sites in the H19 imprinting control region give evidence for insulator function
AU Szabo P.E.; Tang S.-H.E.; Rentsendorj A.; Pfeifer G.P.; Mann J.R.
CS P.E. Szabo, Division of Biology, Research Inst. of the City of Hope, 1450 East Duarte Road, Duarte, CA 91010-3011, United States.
E-mail: pszabo@coh.org
SO Current Biology, ***(18 MAY 2000)*** , 10/10 (607-610), 18 reference(s)
CODEN: CUBLE2 ISSN: 0960-9822
DT Journal; Article
CY United Kingdom
LA English
SL English
AB Parent-of-origin-specific expression of the mouse insulin-like growth factor 2 (Igf2) gene and the closely linked H19 gene are regulated by an intervening 2 kb imprinting control region (ICR), which displays parent-specific differential DNA methylation [1,2]. Four 21 bp repeats are embedded within the ICR and are conserved in the putative ICR of human and rat Igf2 and H19, suggesting that the repeats have a function [3,4]. Here, we report that prominent DNA footprints were found in vivo on the unmethylated maternal ICR at all four 21 bp repeats, demonstrating the presence of ***protein*** binding. The methylated paternal ICR displayed no footprints. Significantly, the maternal-specific footprints were localized to putative binding sites for CTCF, a highly conserved ***zinc*** - ***finger*** DNA-binding ***protein*** with multiple roles in gene regulation including that of ***chromatin*** insulator function [5,6]. These results strongly suggest that the maternal ICR functions as an insulator element in regulating mutually exclusive expression of Igf2 and H19 in cis.

L9 ANSWER 5 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28483729 BIOTECHNO
TI Crystal structure of the BTB domain from PLZF

AU Ahmad K.F.; Engel C.K.; Prive G.G.
CS G.G. Prive, Div. of Molecular/Structural Biol., Ontario Cancer Institute, University of Toronto, 610 University Avenue, Toronto, Ont. M5G 2M9, Canada.
E-mail: prive@oci.utoronto.ca
SO Proceedings of the National Academy of Sciences of the United States of America, ***(13 OCT 1998)*** , 95/21 (12123-12128), 38 reference(s)
CODEN: PNASA6 ISSN: 0027-8424
DT Journal; Conference Article
CY United States
LA English
SL English
AB The BTB domain (also known as the POZ domain) is an evolutionarily conserved ***protein*** - ***protein*** interaction motif found at the N terminus of 5-10% of C.sub.2H.sub.2-type ***zinc*** - ***finger*** transcription factors, as well as in some actin-associated proteins bearing the kelch motif. Many BTB proteins are transcriptional regulators that mediate gene expression through the control of ***chromatin*** conformation. In the human promyelocytic leukemia ***zinc*** ***finger*** (PLZF) ***protein*** , the BTB domain has transcriptional repression activity, directs the ***protein*** to a nuclear punctate pattern, and interacts with components of the histone deacetylase complex. The association of the PLZF BTB domain with the histone deacetylase complex provides a mechanism of linking the transcription factor with enzymatic activities that regulate ***chromatin*** conformation. The crystal structure of the BTB domain of PLZF was determined at 1.9 .ANG. resolution and reveals a tightly intertwined dimer with an extensive hydrophobic interface. Approximately one-quarter of the monomer surface area is involved in the dimer intermolecular contact. These features are typical of obligate homodimers, and we expect the full-length PLZF ***protein*** to exist as a branched transcription factor with two C-terminal DNA- binding regions. A surface-exposed groove lined with conserved amino acids is formed at the dimer interface, suggestive of a peptide- ***binding*** ***site*** . This groove may represent the site of interaction of the PLZF BTB domain with nuclear corepressors or other nuclear proteins.

L9 ANSWER 6 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28471685 BIOTECHNO
TI RP58 associates with condensed ***chromatin*** and mediates a sequence- specific transcriptional repression
AU Aoki K.; Meng G.; Suzuki K.; Takashi T.; Kameoka Y.; Nakahara K.; Ishida R.; Kasai M.
CS M. Kasai, Dept. of Immunology, Natl. Inst. of Infectious Diseases, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162, Japan.
E-mail: masataka@nih.go.jp
SO Journal of Biological Chemistry, ***(09 OCT 1998)*** , 273/41 (26698-26704), 38 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258
DT Journal; Article
CY United States
LA English
SL English
AB An approximately 120-amino acid domain present generally at the NH.sub.2 termini, termed the POZ domain, is highly conserved in various proteins with ***zinc*** ***finger*** DNA binding motifs. We have isolated a novel ***protein*** sharing homology with the POZ domain of a number of ***zinc*** ***finger*** proteins, including the human BCL-6 ***protein*** . By using a ***binding*** ***site*** selection technique (CAST), a high affinity ***binding*** ***site*** of the ***protein*** was determined to be (A/C)ACATCTG(G/T)(A/C), containing the E box core sequence motif. The ***protein*** was shown to repress transcription from a promoter linked to its target sequences and was hence named RP58 (Repressor ***Protein*** with a predicted molecular mass of 58 kDa). Immunogold electron microscopic study revealed that almost all RP58 is localized in condensed ***chromatin*** regions. These observations demonstrate for the first time that a ***protein*** mediating a sequence-specific transcriptional repression associates with highly condensed ***chromatin*** . We suggest that RP58 may be involved in a molecular link between sequence-specific transcriptional repression and the organization of chromosomes in the nucleus.

L9 ANSWER 7 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28335589 BIOTECHNO
TI TIF1.alpha.: A possible link between KRAB ***zinc*** ***finger*** proteins and nuclear receptors
AU Le Douarin B.; You J.; Nielsen A.L.; Chambon P.; Losson R.
CS P. Chambon, Inst. Genet./Biol. Molec./Cellulaire, CNRS/INSERM/ULP, College de France, BP 163, 67404 Illkirch Cedex, France.
SO Journal of Steroid Biochemistry and Molecular Biology, (***1998***), 65/1-6 (43-50), 35 reference(s)
CODEN: JSBBEZ ISSN: 0960-0760
PUI S0960076097001751
DT Journal; Conference Article
CY United Kingdom
LA English
SL English
AB Ligand-induced gene activation by nuclear receptors (NRs) is thought to be mediated by transcriptional intermediary factors (TIFs), that interact with their ligand-dependent AF-2 activating domain. Included in the group of the putative AF-2 TIFs identified so far is TIF1.alpha., a member of a new family of proteins which contains an N-terminal RBCC (RING finger-B boxes-coiled coil) motif and a C-terminal bromodomain preceded by a PHD finger. In addition to these conserved domains present in a number of transcriptional regulatory proteins, TIF1.alpha. was found to contain several ***protein*** - ***protein*** interaction sites. Of these, one specifically interacts with NRs bound to their agonistic ligand and not with NR mutants that are defective in the AF- 2 activity. Immediately adjacent to this 'NR box', TIF1.alpha. contains an interaction site for members of the ***chromatin*** organization modifier (chromo) family, HP1.alpha. and MOD1, which both are heterochromatinic proteins. Finally, TIF1.alpha. also has a ***binding*** ***site*** for KRAB silencing domains of C.sub.2H.sub.2 ***zinc*** ***finger*** proteins. TIF1.beta., another member of the TIF1 gene family, has some interacting partners in common with TIF1.alpha.. TIF1.beta. can interact with HP1.alpha., MOD1 and KRAB domains, but apparently not with NRs. Both TIF1.alpha. and TIF1.beta. repress transcription when fused to a DNA binding domain in transiently transfected mammalian cells. A model discussing the potential function(s) of TIF1s in the control of transcription at the level of the ***chromatin*** template will be presented.

L9 ANSWER 8 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28099683 BIOTECHNO
TI Fusion proteins of the retinoic acid receptor-.alpha. recruit histone deacetylase in promyelocytic leukaemia
AU Grignani F.; De Matteis S.; Nervi C.; Tomassoni L.; Gelmetti V.; Cioce M.; Fanelli M.; Ruthardt M.; Ferrara F.F.; Zamir I.; Seiser C.; Grignani F.; Lazar M.A.; Minucci S.; Pelicci P.G.
CS P.G. Pelicci, Ist. Med. Int. Scienze Oncologiche, Perugia University, 06100 Perugia, Italy.
E-mail: pgpelicci@ieo.cilea.it
SO Nature, ***(19 FEB 1998)*** , 391/6669 (815-818), 29 reference(s)
CODEN: NATUAS ISSN: 0028-0836
DT Journal; Article
CY United Kingdom
LA English
SL English
AB The transforming proteins of acute promyelocytic leukaemias (APL) are fusions of the promyelocytic leukaemia (PML) and the promyelocytic leukaemia ***zinc*** - ***finger*** (PLZF) proteins with retinoic acid receptor-.alpha. (RAR.alpha.). These proteins retain the RAR.alpha. DNA and retinoic acid (RA)-binding domains, and their ability to block haematopoietic differentiation depends on the RAR.alpha. DNA-binding domain. Thus RA-target genes are downstream effectors. However, treatment with RA induces differentiation of leukaemic blast cells and disease remission in PML-RAR.alpha. APLs, whereas PLZF-RAR.alpha. APLs are resistant to RA. Transcriptional regulation by RARs involves modifications of ***chromatin*** by histone deacetylases, which are recruited to RA-target genes by nuclear co- repressors. Here we show that both PML-RAR.alpha. and PLZF-RAR.alpha. fusion proteins recruit the nuclear co-repressor (N-CoR)-histone deacetylase complex through the RAR.alpha. CoR box. PLZF-RAR.alpha. contains a second, RA-resistant ***binding*** ***site*** in the PLZF amino-terminal region. High doses of RA release histone deacetylase activity from PML-RAR.alpha., but not from PLZF-RAR.alpha.. Mutation of the N-CoR ***binding*** ***site*** abolishes the ability of PML-RAR.alpha. to block differentiation, whereas inhibition of histone deacetylase activity switches the transcriptional and biological effects of PLZF-RAR.alpha. from being an inhibitor to an activator of the RA signalling pathway. Therefore, recruitment of histone deacetylase is crucial to the transforming potential of APL fusion proteins, and the different effects of RA on the stability of the PML-RAR.alpha. and PLZF-RAR.alpha. co-repressor complexes determines the differential response of APLs to RA.

L9 ANSWER 9 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1995:25265905 BIOTECHNO
TI A drosophila ***protein*** that imparts directionality on a ***chromatin*** insulator is an enhancer of position-effect variegation
AU Gerasimova T.I.; Gdula D.A.; Gerasimov D.V.; Simonova O.; Corces V.G.
CS Department of Biology, Johns Hopkins University,Baltimore, MD 21218, United States.
SO Cell, (***1995***), 82/4 (587-597)
CODEN: CELLB5 ISSN: 0092-8674
DT Journal; Article
CY United States
LA English
SL English
AB The suppressor of Hairy wing (su(Hw)) ***protein*** inhibits the function of transcriptional enhancers located distally from the promoter with respect to the location of su(Hw)-binding sites. This polarity is due to the ability of the su(Hw)-binding region to form a ***chromatin*** insulator. Mutations in modifier of mdg4 (mod(mdg4)) enhance the effect of su(Hw) by inhibiting the function of enhancers located on both sides of the su(Hw)-binding region. This inhibition results in a variegated expression pattern, and mutations in mod(mdg4)act as classical enhancers of position-effect variegation. The mod(mdg4) and su(Hw) proteins interact with each other. The mod(mdg4) ***protein*** controls the nature of the repressive effect of su(Hw): in the absence of mod(mdg4) ***protein*** , su(Hw) exerts a bidirectional silencing effect, whereas in the presence of mod(mdg4), the silencing effect is transformed into unidirectional repression.

L9 ANSWER 10 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1994:24337786 BIOTECHNO
TI Requirement for RGR1 and SIN4 in RME1-dependent repression in Saccharomyces cerevisiae
AU Covitz P.A.; Song W.; Mitchell A.P.
CS Department of Biological Sciences, Stanford University,Stanford, CA 94305, United States.
SO Genetics, (***1994***), 138/3 (577-586)
CODEN: GENTAE ISSN: 0016-6731
DT Journal; Article
CY United States
LA English
SL English
AB RME1 is a ***zinc*** - ***finger*** ***protein*** homolog that functions as a repressor of the meiotic activator IME1:RME1 is unusual among yeast repressors in two respects: it acts over a considerable distance (2 kbp) and it can activate transcription from a ***binding*** ***site*** separated from its natural flanking region. To identify genes required for RME1 to exert repression, we have selected mutants with improved RME1-dependent activation. One rare mutant was defective in RME1-dependent repression of an artificial reporter gene as well as the native IME1 gene. The mutation permits sporulation of a/a diploids, which express RME1 from its natural promoter, and of a/.alpha. diploids constructed to express RME1 from the GAL1 promoter. The mutation also causes temperature-sensitive growth and a methionine or cysteine requirement. Analysis of a complementing genomic clone indicates that the mutation lies in a known essential gene, RGR1. Prior studies have indicated a functional relationship between RGR1 and SIN4 (also called TSF3); we have found that a sin4 null mutation also causes a defect in RME1-dependent repression and a methionine or cysteine requirement. The rgr1 and sin4 mutations do not cause a reduction of RME1 polypeptide levels. The defect in RME1-dependent repression may result from effects of sin4 and, presumably, rgr1 on ***chromatin*** structure.

L9 ANSWER 11 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1992:22102723 BIOTECHNO
TI The polyhomeotic gene of Drosophila encodes a ***chromatin*** ***protein*** that shares polytene chromosome-binding sites with Polycomb
AU DeCamillis M.; Cheng N.; Pierre D.; Brock H.W.
CS Department of Zoology, University of British Columbia, Vancouver, BC V6T

1Z4, Canada.
SO Genes and Development, (***1992***), 6/2 (223-232)
CODEN: GEDEEP ISSN: 0890-9369
DT Journal; Article
CY United States
LA English
SL English
AB The Polycomb group (PcG) genes in Drosophila melanogaster are required for maintenance of correct spatial expression of homeotic genes, and their products are thought to form either a regulatory network or act as a multimeric complex. Recently, it has been suggested that because of homology between Polycomb (Pc) and Su(var)205, PcG genes encode ***chromatin*** proteins required for the maintenance of a determined state in ***chromatin*** . The polyhomeotic (ph) gene is a member of the PcG of genes. We present DNA sequence of a ph cDNA, which encodes a 169-kD ***protein*** with a single putative ***zinc*** ***finger*** , a serine/threonine-rich region, and has glutamine repeats, suggesting that ph is a DNA-binding ***protein*** . Polyclonal antisera directed against ph ***protein*** bind to .sim.80 sites on polytene chromosomes. Most of these sites appear to be the same as those recognized by antibodies to Pc ***protein*** . ph ***protein*** binds to insertion sites of constructs containing DNA from the bithoraxoid (bxd) region of the Bithorax complex, showing that ph binding to ***chromatin*** is DNA dependent. The same bxd constructs are recognized by Pc ***protein*** , strongly supporting the hypothesis that ph and Pc interact directly.

L9 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:313274 BIOSIS
DN PREV200100313274
TI Acetylation and deacetylation of the promyelocytic leukemia ***zinc*** ***finger*** ***protein*** (PLZF) regulate its ability to function as a transcriptional repressor.
AU Guidez, Fabien [Reprint author]; Ivins, Sarah [Reprint author]; Owen, Gareth [Reprint author]; Hawe, Nicola [Reprint author]; Zelent, Arthur [Reprint author]
CS Leukemia Research Fund Center at the Institute of Cancer Research, Chester Beatty Laboratories, London, UK
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 453a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002
AB The PLZF ***protein*** , originally identified as a fusion with RARalpha in rare cases of all-trans-retinoic acid resistant acute promyelocytic leukemia, is a transcriptional repressor characterised by a C-terminal DNA-binding domain, consisting of nine Kruppel-like zinc fingers, and an N-terminal ***protein*** / ***protein*** interaction domain, the POZ domain. Expression studies of PLZF throughout hematopoiesis, as well as its over-expression in hematopoietic progenitor cells, suggest that in addition to being involved in leukemogenesis, PLZF plays an important role in regulating growth and differentiation of normal myeloid precursors. Previous work has also shown that PLZF can recruit components of the nuclear receptor co-repressor complexes, such as N-CoR and histone deacetylase (HDAC) through the POZ domain, raising the possibility that effects of PLZF on gene transcription are mediated through an HDAC dependent mechanism. We now show directly that transcriptional repression by PLZF correlates with deacetylation of more histones present in ***chromatin*** surrounding its DNA ***binding*** ***site*** . Furthermore, the ability of PLZF to repress transcription and cause deacetylation of histone H4 in vivo is highly dependent on acetylation of lysine residues in its C-terminal ***zinc*** - ***finger*** . Acetylation of PLZF is mediated by acetyltransferase activity of the p300 ***protein*** (but not SRC-1 or p/CAF), with which PLZF associates in vitro and in vivo. As expected from the above results, transient co-expression of p300, but not its histone acetyltransferase domain deficient mutant, enhances transcriptional repression by PLZF. Mutating the target lysine residues to arginines, which cannot be acetylated, completely abolishes the ability of PLZF to cause histone deacetylation and repress transcription in vivo. Furthermore, co-expression of HDAC3 (but not HDAC1), which also interacts with and deacetylates PLZF in vitro, relieves repression byt the wild type PLZF ***protein*** . Taken together, our results indicate that a histone deacetylase dependent transcriptional repressor, can be regulated through a cycle of acetylation/deacetylation of lysine residues in its most C-terminal ***zinc*** - ***finger*** . The indirect effect of acetylation and deacetylation of PLZF thus serves to stimulate and inhibit its ability to repress target gene transcription.

L9 ANSWER 13 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:312498 BIOSIS
DN PREV200100312498
TI Identification of the ***protein*** 4.2 gene as a direct target of the TAL1/SCL transcription factor in differentiating murine erythroleukemia cells.
AU Xu, Zhixiong [Reprint author]; Huang, Suming [Reprint author]; Chang, Long-Sheng; Brandt, Stephen J. [Reprint author]
CS Medicine, Vanderbilt University Medical Center, Nashville, TN, USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 497a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002
AB The TAL1/SCL gene, originally identified through its involvement by a recurrent chromosomal translocation, encodes a basic helix-loop-helix (bHLH) transcription factor with an essential role in embryonic hematopoiesis. Although TAL1 likely alters the transcription of a specific set of genes, no targets have been definitively identified. ***Binding*** ***site*** selection assays in erythroid cells have suggested that TAL1 contributes to a multiprotein DNA-binding complex that also includes the ***zinc*** ***finger*** ***protein*** GATA-1 and has a preference for a bipartite sequence motif containing an E box (bHLH ***protein*** ***binding*** ***site***) and a GATA site. We identified two such E box-GATA elements in the proximal promoter of the

murine ***protein*** 4.2 gene and investigated whether this gene could be a target for these transcription factors. First, a TAL1- and GATA-1-containing complex was detected by gel shift analysis using both E box-GATA elements in the promoter as probes. Further, an increase in these DNA-binding activities was observed with DMSO-induced differentiation of murine erythroleukemia (MEL) cells, concomitant with an increase in expression of endogenous ***protein*** 4.2 mRNA. Cold competitor studies and DNA-binding assays with mutated probes indicated the requirement for both E box and GATA sites in these elements for formation of these binding complexes. In addition, reporter assays showed that DMSO-induced promoter activity decreased by approximately 75% and 90%, respectively, with mutation of either E box or GATA site, suggesting that both elements contribute to promoter activity and that the E box and GATA sites in these elements are both required for maximal induction of ***protein*** 4.2 promoter activity during MEL cell differentiation.

An expression vector for TAL1 increased promoter activity in reporter assays when cotransfected with its DNA-binding partner E47, GATA-1, and the LIM domain ***protein*** LMO2. Finally, an increase in endogenous ***protein*** 4.2 gene expression and in E box-GATA DNA-binding activity was observed when TAL1 was overexpressed in cells, a decrease in both was observed when a binding-defective TAL1 dominant negative mutant was introduced, and direct evidence for TAL1 occupancy of the promoter in cells was obtained by ***chromatin*** immunoprecipitation analysis. In sum, these data establish the ***protein*** 4.2 gene as a physiologic target of a TAL1- and GATA-1-containing complex in differentiating murine erythroleukemia cells.

L9 ANSWER 14 OF 15 LIFESCI COPYRIGHT 2004 CSA on STN
AN 1999:1250 LIFESCI
TI TIF1 alpha : A possible link between KRAB ***zinc*** ***finger*** proteins and nuclear receptors
AU Le Douarin, B.; You, J.; Nielsen, A.L.; Chambon, P.*; Losson, R.
CS Institut de Genetique et de Biologie Moleculaire et Cellulaire, CNRS/INSERM/ULP, College de France, BP 163, 67404 Illkirch Cedex, France
SO J. Steroid Biochem. Mol. Biol., (***19980400***) vol. 65, no. 1-6, pp. 1-6.
ISSN: 0960-0760.
DT Journal
FS N
LA English
SL English
AB Ligand-induced gene activation by nuclear receptors (NRs) is thought to be mediated by transcriptional intermediary factors (TIFs), that interact with their ligand-dependent AF-2 activating domain. Included in the group of the putative AF-2 TIFs identified so far is TIF1 alpha , a member of a new family of proteins which contains an N-terminal RBCC (RING finger-B boxes-coiled coil) motif and a C-terminal bromodomain preceded by a PHD finger. In addition to these conserved domains present in a number of transcriptional regulatory proteins, TIF1 alpha was found to contain several ***protein*** - ***protein*** interaction sites. Of these, one specifically interacts with NRs bound to their agonistic ligand and not with NR mutants that are defective in the AF-2 activity. Immediately adjacent to this 'NR box', TIF1 alpha contains an interaction site for members of the ***chromatin*** organization modifier (chromo) family, HP1 alpha and MOD1, which both are heterochromatinic proteins. Finally, TIF1 alpha also has a ***binding*** ***site*** for KRAB silencing domains of C sub(2)H sub(2) ***zinc*** ***finger*** proteins. TIF1 beta , another member of the TIF1 gene family, has some interacting partners in common with TIF1 alpha . TIF1 beta can interact with HP1 alpha , MOD1 and KRAB domains, but apparently not with NRs. Both TIF1 alpha and TIF1 beta repress transcription when fused to a DNA binding domain in transiently transfected mammalian cells. A model discussing the potential function(s) of TIF1s in the control of transcription at the level of the ***chromatin*** template will be presented.

L9 ANSWER 15 OF 15 MEDLINE on STN
AN 97185912 MEDLINE
DN PubMed ID: 9033593
TI The solution structure of a specific GAGA factor-DNA complex reveals a modular binding mode.
CM Comment in: Nat Struct Biol. 1997 Feb;4(2):87-9. PubMed ID: 9033581
AU Omichinski J G; Pedone P V; Felsenfeld G; Gronenborn A M; Clore G M
CS Laboratories of Chemical Physics, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, Maryland 20892-0520, USA.
SO Nature structural biology, ***(1997 Feb)*** 4 (2) 122-32.
Journal code: 9421566. ISSN: 1072-8368.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199703
ED Entered STN: 19970327
Last Updated on STN: 19980206
Entered Medline: 19970320
AB The structure of a complex between the DNA binding domain of the GAGA factor (GAGA-DBD) and an oligonucleotide containing its GAGAG consensus ***binding*** ***site*** has been determined by nuclear magnetic resonance spectroscopy. The GAGA-DBD comprises a single classical Cys2-His2 ***zinc*** ***finger*** core, and an N-terminal extension containing two highly basic regions, BR1 and BR2. The ***zinc*** ***finger*** core binds in the major groove and recognizes the first three GAG bases of the consensus in a manner similar to that seen in other classical ***zinc*** ***finger*** -DNA complexes. Unlike the latter, which require tandem ***zinc*** ***finger*** repeats with a minimum of two units for high affinity binding, the GAGA-DBD makes use of only a single finger complemented by BR1 and BR2. BR2 forms a helix that interacts in the major groove recognizing the last G of the consensus, while BR1 wraps around the DNA in the minor groove and recognizes the A in the fourth position of the consensus. The implications of the structure of the GAGA-DBD-DNA complex for ***chromatin*** remodelling are discussed.

=> log h

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